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# Anaesthetic Gases and Vapours

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for Occupational
Health and Safety

# Anaesthetic Gases and Vapours

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# ANAESTHETIC GASES AND VAPOURS

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## Preface

This document has been prepared for a readership consisting primarily of those people regularly exposed to anaesthetic gases in the course of their daily work, such as operating room and recovery room nurses, anaesthetists and dentists. The aim is to present a straightforward summary of the findings from an extensive research effort into the possible health effects from exposure to anaesthetic gases and the control strategies available to minimize the exposure.

## Introduction

Sir Humphry Davy in 1800 noted the anaesthetic effect of nitrous oxide and suggested that it might prove useful in surgery. But, it was not until 1847 that Horace Wells attempted to use it as an inhalation anaesthetic for dental surgery. Around that same time two American surgeons, A.V. Long and T.G. Morton, successfully demonstrated the use of diethyl ether for general anaesthesia, while the English surgeon, Sir J.Y. Simpson, introduced the use of chloroform for relieving the pain of childbirth.

Other agents were added over time to the arsenal of anaesthetics, including ethyl chloride, trichloroethylene, and cyclopropane. While nitrous oxide remains the most widely used inhalation anaesthetic today, most of the other early anaesthetic agents have been replaced in the last thirty years by a group of non-flammable, non-explosive, halogenated agents. Those that have found widespread use are listed below together with their commonly used names.

| Chemical Name | Common Names |
|---|---|
| 2-bromo-2-chloro-1,1,1-trifluoroethane | halothane, fluothane |
| 2,2-dichloro-1,1-difluoroethyl-methylether | methoxyflurane, penthrane |
| 2-chloro-1,1,2-trifluoroethyl-difluoromethylether | enflurane, ethrane |
| 2,2,2-trifluorochloroethyl-difluoromethylether | isoflurane, forane |
| 2,2,2-trifluoroethylvinylether | fluoroxene, fluoromar |

Exposure to anaesthetic gases and vapours is experienced by surgeons, nurses and anaesthetists in the operating room. Until recent years, they were exposed to elevated concentrations since it was common practice to vent excess gas and vapour directly into the room. Over the last ten to fifteen years, progress has been made towards reduction of this exposure by the introduction of

**Introduction**
*continued*

systems to remove waste gases (scavenging systems). Exposure is also of concern to workers in recovery rooms, delivery rooms, cryotherapy rooms, dental operatories, veterinary operatories, and animal research facilities.

The possible adverse health effects resulting from this exposure have been extensively studied. By far the greatest attention has been paid to adverse effects upon pregnancy outcome, but other effects have been reported or investigated including effects upon task performance, systemic effects upon the liver and kidney, and carcinogenic effects. These studies have been concerned mainly with exposure to nitrous oxide and halothane.

This document will be concerned with this body of evidence and will also deal with the techniques that have been recommended to reduce exposure.

**Evidence for adverse reproductive effects**

Epidemiological studies

In 1967, a Russian researcher, Vaisman, (as cited in reference 1) conducted a questionnaire survey of 193 male and 110 female anaesthetists and found a high incidence of headache, excessive fatigue, irritability and nausea. Even more noteworthy, 18 of 31 pregnancies among the female anaesthetists ended in miscarriage. In addition to exposure to anaesthetic gases, at that time mainly diethylether, Vaisman suggested that this excessive rate of spontaneous abortion might arise, at least in part, from stressful working conditions such as long and irregular hours of work, and the need for constant vigilance.

Vaisman's report spawned a number of studies in several countries, all aimed at determining whether occupational exposure to anaesthetic gases poses a reproductive hazard. All but one of the studies had essentially the same design. One group of workers (including anaesthetists and operating room nurses) was identified as the exposed group. Another group (including paediatricians and surgical ward nurses) who did not work in areas with exposure to anaesthetic gases was identified as the control. Questionnaires were distributed, usually by mail, to the two groups. Responses to questions on pregnancy outcome in the two groups were compared. Differences in rates of adverse pregnancy outcome in the two groups were then tested for statistical significance, to determine the probability that any difference may have arisen by chance.

The results of these studies (see table 1, p. 4 and table 2, p. 5) have not clearly established that there is a reproductive hazard associated with work in the operating room. Several of the early studies following Vaisman's report supported the conclusions that operating room work was associated with increased spontaneous

**Epidemiological studies** *continued*

abortion and noted a trend, usually not statistically significant, towards increased congenital abnormality. The risk ratios in these studies were consistently less dramatic than in the original Russian report but still great enough to be of concern.

This additional evidence of adverse effects led the American Society of Anaesthesiologists (ASA) to mount a large-scale, nation-wide study involving close to 50,000 operating room personnel including anaesthetists, nurse anaesthetists, operating room nurses and operating room technicians[9]. The control group consisted of paediatricians and nurses not working in the operating theatre. The study found a significantly higher rate of spontaneous abortions among female anaesthetists when compared with paediatricians; a significantly higher rate of spontaneous abortions among the operating room nurses and technicians compared with non-operating room nurses. However, there was no statistically significant increased risk for nurse anaesthetists, nor was there any evidence for increased risk of spontaneous abortion in the wives of exposed males.

**Table 1**
**Spontaneous Abortion in Females Exposed to Anaesthetic Gases**

| Authors | Year | Occupation of Exposed Group | Occupation of Controls | Exposed Group | | Control | | Significance |
|---|---|---|---|---|---|---|---|---|
| | | | | No. of Pregnancies | % ending in abortion | No. of Pregnancies | % ending in abortion | |
| Vaisman | 1967 | Anaesthesiologist | ------- | 31 | 58 | ------ | ------ | ------ |
| Askrog & Harvald | 1970 | Nurse | ------- | 229 | 17 | (85) | (12) | N.S. |
| Cohen et al | 1971 | O.R. Nurse Anaesthetist | General Duty Nurse | 36 | 28 | 34 | 9 | <0.05 |
| | | | Other Physicians | 37 | 38 | 58 | 10 | <0.01 |
| Knill-Jones et al | 1972 | Anaesthetist | Other Physicians | 737 | 18 | 2150 | 15 | <0.05 |
| Rosenburg & Kirves | 1973 | Nurse O.R | Nurse Casualty, I.C. | 257 | 20 | 150 | 11 | <0.05 |
| Cohen et al | 1974 | Nurse Anaesthetist | Nurse (Non O.R.) | 1826 | 17 | 1948 | 15 | N.S. |
| | | Nurse/Technician Anaesthetist | Nurse (Non O.R.) | 2781 | 20 | 1948 | 15 | <0.001 |
| | | | Paediatricians | 468 | 17 | 308 | 9 | <0.01 |
| Knill-Jones et al | 1975 | Anaesthetist | Other Physicians | 523 | 16 | 7296 | 11 | <0.01 |
| Pharoah | 1977 | Anaesthetist | Other Physicians | 670 | 14 | 8374 | 13 | N.S. |
| Rosenburg et al | 1978 | Anaesthetist | Paediatricians & Paediatricians Wives | 86 | 9 | 266 | 13 | N.S. |
| Tomlin | 1979 | Anaesthetist | ------- | 58 | 29 | (102) | (10) | <0.05 |
| Cohen et al | 1980 | Dental Assistant using Anaesthetics | Dental Assistants not using Anaesthetics | 400 | 19 | 3184 | 8 | <0.01 |
| Lauwerys et al | 1981 | Anaesthetist Nurse O.R. | Other Physicians Other Nurses | 259 | 8 | 1519 | 6 | N.S. |
| Axelsson, Rylander | 1982 | Operating Room and Others | Medical Ward | 185 | 12 | 470 | 9 | N.S. |

**Table 2**
**Malformations of Offspring of Exposed Females**

| Authors | Year | Occupation of Exposed Group | Occupation of Controls | Exposed Group No. of infants born | Exposed Group % malformed | Control No. of infants born | Control % malformed | Significance |
|---|---|---|---|---|---|---|---|---|
| Askrog & Harvald | 1970 | Nurse | ------- | 185 | 0.5 | (75) | (0) | N.S. |
| Knill-Jones et al | 1972 | Anaesthetist | Other Physicians | 893 | 3.0 | 1835 | 3.2 | N.S. |
| Rosenburg & Kirves | 1973 | Nurse O.R. | Nurse Casualty & I.C. | 207 | 0 | 133 | 0 | N.S. |
| Corbett et al | 1974 | Nurse | ------- | 434 | 8.8 | (261) | 3.8 | <0.05 |
| Cohen et al | 1974 | Nurse | Nurse (Non O.R.) | 1480 | 9.6 | 1629 | 7.6 | <0.05 |
| | | Nurse/Technician | Nurse (Non O.R.) | 210 | 7.7 | 1629 | 7.6 | N.S. |
| | | Anaesthetist | Paediatricians | 384 | 5.9 | 276 | 3.0 | N.S. |
| Knill-Jones et al | 1975 | Anaesthetist | Other Physicians | 438 | 1.6 | 6442 | 1.1 | N.S. |
| Pharoah et al | 1977 | Anaesthetist | Other Physicians | 578 | 2.8 | 7317 | 1.8 | N.S. |
| Tomlin | 1979 | Anaesthetist | ------- | 277 | 9.4 | 92 | 4.3 | N.S. |
| Cohen et al | 1980 | Dental Assistant using Anaesthetics | Dental Assistant not using Anaesthetics | 316 | 5.2 | 2882 | 3.6 | N.S. |

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Epidemiological studies *continued*

The ASA study noted a trend towards increased risk of congenital abnormality in the exposed groups, but this was only significant for the nurse anaesthetist group. If exposure to anaesthetic agents was responsible for adverse reproductive effect, it would be expected that the adverse effect be observed consistently in all of the exposed groups. The inconsistencies among the exposed groups in this study make the interpretation of the findings difficult.

The ASA study was followed by a number of other investigations, generally smaller in scope, which have given contradictory results (see Table 1). The more recent studies have failed to find any association between work in operating theatres and spontaneous abortion[12,14]. With the exception of the one elevated rate of congenital abnormality in the offspring of nurse anaethetists reported in the ASA national study, none of the subsequent investigations has been able to demonstrate a statistically significant association of this particular problem with work in the operating theatre.

The validity of the results of these epidemiological investigations has been questioned because of the many limitations inherent in studies with this type of design. These limitations include potential biases in the data resulting from inadequate response rates, different response rates between the exposed and control groups, difficulties in obtaining good comparison groups, and lack of exposure data for anaesthetic gases. Most important are problems produced by differences in the ability of exposed and control groups to recall events related to their past pregnancies sometimes from 10 to 30 years prior to the administration of the questionnaire.

This last problem was demonstrated clearly in a Swedish study[12]. The researchers compared the answers to a postal questionnaire on spontaneous abortion in female personnel exposed to anaesthetic gases and a control group not having such exposure. The investigation was also conducted using hospital records instead of the questionnaire to obtain the data. They found that all of the miscarriages in the group exposed to anaesthetic agents were reported in the questionnaire, while one third of all miscarriages occurring in the control population were not reported.

Recently, a Finnish study used a different research design in an attempt to overcome the problems encountered in the previous investigations[14]. The study design was that of case control. Using hospital records and a register of congenital malformations, nurses who had spontaneous abortions or malformed children were selected. These nurses formed the case group. A control group consisted of nurses who had normal births. The nurses in both case and control groups were classified according to whether or not they had daily exposure to anaesthetic gases during the first trimester of pregnancy. The ratio of those who had exposure to

Epidemiological studies *continued*

those who did not was not statistically different in the cases and the controls. This study suffered from the possibility of exposure misclassification based on questionnaire responses by head nurses. The possibility also exists that many of the nurses classified as exposed may have only had limited exposure to anaesthetic gases because the study period encompassed the time that control devices were being introduced into operating rooms for the removal of waste anaesthetic gases and vapours.

Animal studies

Experimental animal studies have been conducted in an attempt to determine whether exposure to anaesthetic gases and vapours is capable of producing miscarriage or congenital abnormalities. The majority of these studies focussed upon nitrous oxide and halothane. (see table 3, p. 8)

Early experiments demonstrated that both nitrous oxide and halothane were capable of producing fetal resorption (animal equivalent to spontaneous abortion) and congenital abnormalities in the small animal species used. However, the concentrations employed in these experiments were at levels close to or above those required to produce anaesthesia.

Animal studies conducted at concentrations more closely approaching those encountered in operating theatres without scavenging devices have consistently failed to demonstrate any such effect for halothane or for other halogenated hydrocarbons. The results for nitrous oxide exposure have been inconsistent. The majority of the studies have been negative, but Viera and co-workers, in a series of experiments, succeeded in producing excessive resorptions in rats exposed to nitrous oxide at 1000 ppm or more[20]. These results agreed with earlier work by Corbett, who also produced resorptions in animals exposed at or in excess of 1000 ppm[18]. These experiments give some support to the idea that operating rooms not equipped with scavenging systems may have produced exposures capable of causing spontaneous abortion.

**Table 3**
**Animal Data**

| Author | Species | Agent/Concentration | Resorption | Defects |
|---|---|---|---|---|
| Fink et al. 1967 | Rat | $N_2O$/500,000 PPM | Yes | Yes |
| Shepard & Fink, 1958 | Rat | $N_2O$/700,000 PPM | --- | Yes |
| Dussard et al. 1974 | Hamster | $N_2O$/600,000 PPM and Halothane/6,000 PPM | Yes | --- |
| Basford & Fink 1968 | | Halothame/6,000 PPM | --- | Yes |
| Smith et al. 1971 | Mice | Halothame/10,000 or 15,000 PPM | --- | Yes |
| Corbett et al. 1973 | Rat | Nitrousoxide/100 PPM<br>Nitrousoxide/1,000 PPM<br>Nitrousoxide/15,000 PPM | No<br>Yes<br>Yes | ---<br>---<br>--- |
| Lansdown et al. 1978 | Rat | Halothane/50 PPM<br>/100 PPM<br>/200 PPM<br>/800 PPM<br>/1,600 PPM<br>/13,200 PPM | No | No |
| Bruce et al. 1973 | Mice | Halothane/16 PPM | No | No |
| Wharton et al. 1978 | Mice | Halothane/500 PPM<br>/1,000 PPM<br>/3,000 PPM<br>/10,000 PPM | No | --- |
| Popova et al. 1979 | Mice | Halothane/9 PPM | Yes | --- |
| Wharton et al. 1979 | Mice | Halothane/500 PPM<br>/1,000 PPM<br>/3,000 PPM<br>/10,000 PPM | ---<br>---<br>---<br>--- | No<br>No<br>Yes<br>Yes |
| Coate et al. 1979 | Rat | $N_2O$/50 PPM & halothane/1 PPM<br>$N_2O$/500 PPM & Halothane/ 10 PPM | No<br>No | Yes<br>Yes |
| Viera 1979 | Rat | $N_2O$/5,000 PPM | Yes | Yes |
| Viera et al. 1980 | Rat | $N_2O$/1,000 PPM<br>$N_2O$/500 PPM<br>$N_2O$/250 PPM | Yes<br>No<br>No | Yes<br>No<br>No |
| Mazze et al. 1982 | Mice | $N_2O$/5,000-500,000 PPM | No | No |
| Viera et al. 1983 | Rat | $N_2O$/250-5,000 PPM intermittently | No | No |

## Evidence for other effects

Effects other than reproductive effects have been reported from exposure to anaesthetic gases and vapours. Some of the more important will be dealt with in this section.

### Carcinogenicity

In 1973, Corbett reported in a small study that cancer in a group of Michigan nurse anaesthetists occurred at a higher than expected frequency[15]. Later, the major ASA study described above, found that there was an increased occurrence of cancer in female nurse anaesthetists and increases of borderline significance in female anaesthetists and operating room nurses. No such increase was found among the male population of these three professions.

Ferstandig[4], in a close examination of Corbett's data, showed that the rate was high for only one year of the study period and that when all the available data were considered there was in fact no significant difference between the cancer rate experienced by the nurse anaesthetists and that of the control group. The ASA study, as noted above, has many limitations making the reliability of the findings questionable. Nevertheless, these studies raise the possibility that anaesthetic gases and vapours could be carcinogenic.

There have been a number of studies of causes of death among anaesthetists, the professional group most exposed to these agents. All have failed to show any unusual mortality rate for any form of cancer. The most recent study found that anesthesiologists practicing in the earlier part of this century had lower death rates and were less likely to die of cancer than the general white male population[8]. The death rates through the years 1930-46 were similar to those during 1947-56, but greater than that for 1957-71, suggesting that the introduction of the modern fluorinated anaesthetics in the mid-1950's is not associated with any major health hazard.

In animal studies, the anaesthetic agents chloroform and trichloroethylene have been shown to be carcinogenic at very high concentrations. However, these materials are not used today.

Corbett found that mice exposed to isoflurane developed lung cancer and liver cancer at rates which were greater than control groups, although not significantly so[21]. The study was methodologically defective and was repeated by Eger and co-workers[22]. They found no evidence for carcinogenicity of isoflurane. In the same set of experiments they also found no carcinogenic potential for enflurane, halothane, methoxyflurane, and nitrous oxide. However, these experiments were terminated earlier than the 24 months recommended in the National Cancer Institute protocol. Experiments by other researchers also found no carcinogenic effects of halothane in exposed animals.

**Carcinogenicity** *continued*

All of the commonly used modern inhalation anaesthetics have been tested for mutagenicity in bacterial systems (mutagenicity tests are performed primarily as screening tests for carcinogens since chemicals which are carcinogenic are usually mutagenic also). All were negative in most test systems except for fluroxene[23].

**Effects upon the liver and kidney**

The ASA national study reported that liver disease occurred more frequently in both males and females exposed to anaesthetic agents. The correlation of work in operating theatres and reported kidney disease was less strong. Similar findings were obtained in a questionnaire study of dentists and dental assistants exposed to inhalation anaesthetics[11]. A study in England also gave evidence of increased liver disorders among anaesthetists compared with other physicians[6]. On the other hand, mortality studies have not shown any increased mortality rates from either liver or kidney disease.

There have been some case reports of liver dysfunction among patients anaesthetized with nitrous oxide/halothane mixtures and in anaesthetists exposed to halothane. Kidney effects have been noted in patients anaesthetized with methoxyflurane. Such effects have not been reported for enflurane anaesthesia.

Animal experiments demonstrated liver and kidney damage at high (anaesthetic) doses of halothane. Chronic exposure to subanaesthetic, but still high, concentrations of halothane produced liver damage; isoflurane and diethylether did not. Nitrous oxide was also shown to be without effect. Inconsistent results were produced with enflurane while fluroxene was very toxic even at 30 ppm. In these experiments there was no adverse effects produced in the kidney.

In a study of delivery ward personnel exposed to methoxyflurane, some alterations in liver and kidney function were observed three days after exposure.

**Effects upon the nervous system**

Habitual use of nitrous oxide has been linked to damage of the peripheral nervous system. However, such damage has not been reported in man or animals as a result of nitrous oxide exposure at occupational levels.

Effects upon the central nervous system (CNS) have been evaluated by the use of a battery of tests designed to measure performance, including tests of perceptual motor skills, short-term memory, and certain aspects of intelligence. While it is clear that exposure to anaesthetics at sufficient concentrations will produce a decrement in performance, the question of the threshold at which they begin to affect performance remains controversial.

**Effects upon the nervous system** *continued*

Bruce and co-workers found that student volunteers exposed to various concentrations of nitrous oxide (500 ppm and 50 ppm) and nitrous oxide and halothane (500 and 10 ppm and 50 and 1 ppm) had measurable decrements in performance on one or more of these tests[26]. However, attempts to duplicate these findings in four separate laboratories have all failed[27]. One study indicates that the threshold for performance decrement following nitrous oxide exposures is between 8 and 12%, (80,000 and 120,000 ppm).

In an attempt to examine the effects of exposure to anaesthetic agents one study examined the driving skills of operating room nurses exposed to nitrous oxide and halothane compared to ward nurses not exposed to anaesthetic gases, and found no differences.

No studies of central nervous system effects from long term exposure to waste anaesthetic gases have been found.

**Conclusions from evidence**

It appears from the evidence available that acute exposure to occupational levels of anaesthetic gases does not noticeably affect the central nervous system. But the effects of chronic long-term exposure do require further investigation.

There is evidence of liver and kidney damage from exposure to anaesthetic agents, but only at concentrations well in excess of those encountered in occupational exposure in the operating room. Mortality studies on anaesthetists have not implicated either liver or kidney in increased mortality when compared with other physicians. It appears unlikely that these agents would present a problem in this context under exposure conditions in modern operating rooms.

On the basis of current evidence, modern anaesthetic agents can not be considered to be carcinogenic. But further research by animal assay is probably warranted, since the investigations have not been extensive.

The evidence that anaesthetic agents can cause adverse effects upon pregnancy outcome is largely epidemiological. The studies conducted so far have design limitations that make interpretation difficult. It cannot be claimed that they have produced any reliable evidence of a reproductive hazard in the operating theatre. Some of the studies do suggest an excess of spontaneous abortions in exposed women. There is limited evidence from animal experiments that exposure to nitrous oxide at levels which were not uncommon in unscavenged theatres can produce such effects.

It is worth noting that, even if the epidemiological evidence for adverse effects had not been equivocal, the results would still not have directly implicated anaesthetic gases. All of the studies lacked

**Conclusions from evidence**
*continued*

quantitative data on exposure to these agents and were thus incapable of demonstrating a cause/effect relation. This shortcoming is important, since there are other agents to which operating theatre personnel are exposed which could contribute to if not cause reproductive problems (such as x-rays, methyl methacrylate and stress).

In view of the possibilities that anaesthetic agents adversely affect (a) reproductive outcome, and (b) liver and kidney at high concentrations, combined with the lack of information on CNS effects from long term exposure, the prudent course of action is to introduce controls to reduce exposures. In Canada and elsewhere this has been systematically taking place.

**Typical exposure levels**

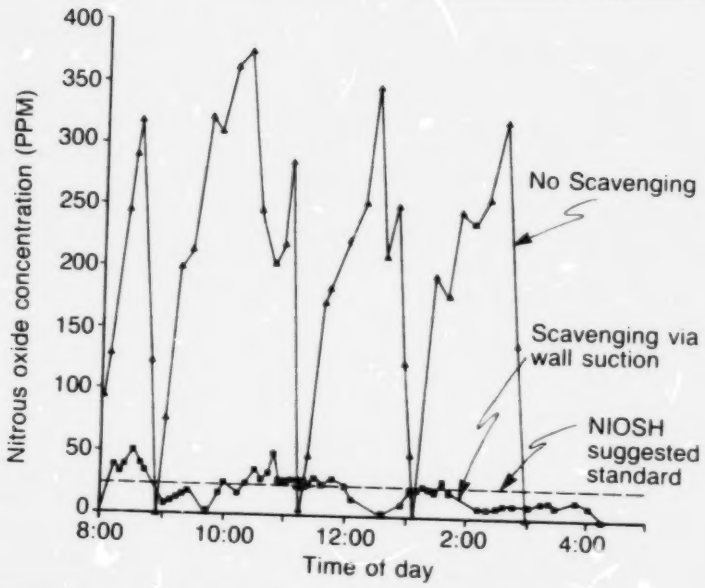
Many studies have been conducted to measure the concentration of anaesthetic gases and vapours in operating theatres. Theatres equipped with control devices (scavenging devices) and others not so equipped have been evaluated.

The airborne levels of anaesthetic agents in the operating room depend upon many factors including the breathing circuit used, the flow rates employed, the anaesthetic technique, the presence of leaks in equipment, the use of a scavenging device, and the capacity of the general ventilation system supplying and exhausting air from the operating room.

In operating rooms without scavenging devices, nitrous oxide concentrations have been reported to range from 0-10,000 ppm, but are typically below 1000 ppm. Halothane concentrations have been reported between 0 and 200 ppm, with measurements typically being less than 20 ppm. In operating rooms with scavenging devices, nitrous oxide levels of 0-600 ppm and halothane concentrations of 0-8 ppm have been reported. Levels of below 35 ppm for nitrous oxide and below 2 ppm for halothane appear to be readily achievable. Figure 1 illustrates the degree of improvement that can be achieved through the use of scavenging devices[28].

**Figure 1**

Variation in $N_2O$ concentration over operating table (two typical days, one with scavenging, one without)




## Exposure standards and guidelines

The National Institute for Occupational Safety and Health in the United States suggested, in 1976, that exposure to nitrous oxide be limited to 25 ppm and exposure to halogenated anaesthetics be limited to 2 ppm. This suggestion was based upon what was considered achievable rather than defining a level known or considered to be "safe." The use of this guideline has continued until today in many jurisdictions, probably because there is little more reliable data on harmful effects now than there was at that time.

In Sweden and West Germany, exposure to halothane has been limited to 5 ppm. One report suggests that in Denmark, Norway and Sweden, hygienic standards for nitrous oxide levels in the operating theatre have been set at 100 ppm and in Yugoslavia at 5 ppm, apparently based upon the effects on performance described in Section 3.

Most Canadian provinces base their standards on guidelines suggested by the American Conference of Governmental Industrial Hygienists. This body has not to date included any anaesthetic agents in its guidelines, but has proposed in its list of intended changes that halothane exposure be limited to 50 ppm and enflurane to 75 ppm.

At present there are no legislated standards for anaesthetic gas exposure in Canada. In most juridictions, the NIOSH recommendations are used as guidelines. Local occupational health and safety authorities should be contacted to determine what they are recommending for their jurisdiction.

## Control Strategies

Control of exposure to anaesthetic gases and vapours is achieved by:

1. effective scavenging devices for the removal of excess and exhaled gases,
2. maintenance procedures designed to detect and eliminate leaks in the anaesthesia delivery equipment, and
3. good technique on the part of the anaesthetist.

The effectiveness of these efforts to control the exposure can be evaluated by routine monitoring of contaminant levels.

### Scavenging Systems

Scavenging systems have three components: a collection device (scavenging device) to collect the waste anaesthetic gases in the breathing circuit and ventilator; a disposal system to carry the collected gases from the operating room; and a device for ensuring that excessive negative or positive pressures in the system, which could adversely affect the patient, do not occur. Manufacturers of anaesthetic machines and ventilators provide scavenging valves on new equipment. Old equipment can be modified by the manufacturer or a technician.

The scavenging device causes gases from the adjustable pressure-limiting valve in the patient breathing circuit or from the automatic ventilator to be routed into a disposal system. There are active and passive scavenging systems. Active scavenging systems utilize the hospital central vacuum system, while passive scavenging systems use either the non-recirculating room ventilation system or a dedicated exhaust system.

For passive systems the use of a dedicated exhaust system serving only for the removal of anaesthetic gases would be ideal. However, these are usually only encountered in new facilities because of high retrofit costs. More commonly, passive systems use the exhaust duct of the ventilation system to provide a simple and effective method of removal of gases. The potential problem of blockage leading to excessive positive pressure is overcome by a positive pressure relief valve. While only minimal negative pressures are expected in the disposal system, some of the literature indicates that, in some instances, negative pressures can be of concern. A device for relief of negative pressure is recommended unless it can be ensured that negative pressures cannot be created. A bag for collecting vented gases in short term excess of the disposal system capacity should be included in the circuit.

The use of the central vacuum system for removal of waste anaesthetic gases requires the use of an appropriate interface device for relief of negative pressure. It has been suggested that scavengers

Scavenging systems *continued*

that vent into the vacuum system require a minimum vacuum flow of 10 litres/minute[31]. If the vacuum system is not a dedicated system, then the ability of the vacuum system to meet all demands from surgery and anaesthesia should be verified.

A Canadian study could not determine any difference in effectiveness between passive and active scavenging systems[29]. The decision as to which to install must be made on the basis of local factors such as the lengths of tubing required for the system, the capacity of the vacuum system and the need for air recirculation.

Schematic diagrams of some systems are shown in Figures 2 and 3.

**Figure 2** Schematic of typical HVAC scavenger

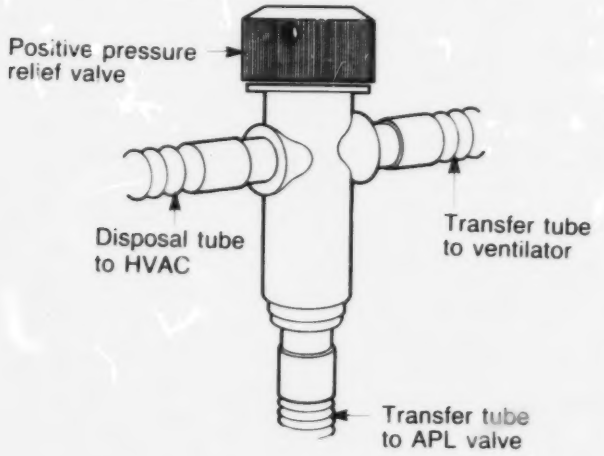
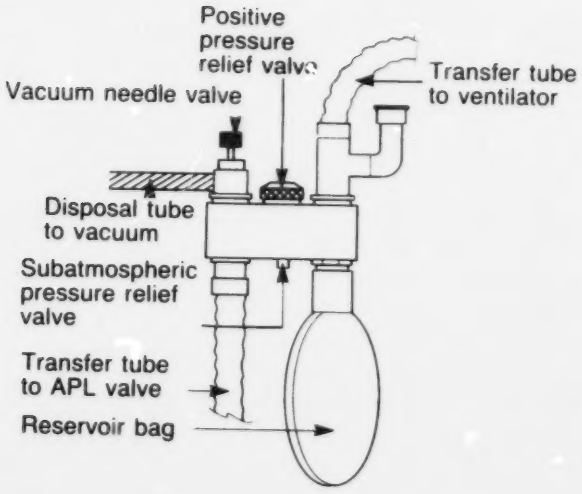



**Figure 3** Schematic of typical Vacuum scavenger



## Equipment maintenance

The scavenging system can only remove those waste gases that are collected from the breathing circuits. Elevated levels of anaesthetic gases and vapours can still occur in the theatre from the development of leaks in the anaesthetic delivery system. High pressure leaks occur in the nitrous oxide delivery system usually at intermediate connections and at the point of connection to the machine. Even small leaks here can lead to a considerable increase in the level of this contaminant. In the low pressure system, leaks occur at connections in hoses, in the ventilator, at the pop-off valves and in the carbon dioxide absorption circle units. Routine procedures are therefore required for the detection of leaks and their correction. Figures 4 and 5 show some of the common high and low pressure leakage points. One study carried out in Alberta operating theatres has shown that hospitals with maintenance procedures aimed at leak detection and correction, as well as patient safety, consistently had lower levels of contamination in their operating rooms than found in rooms without such procedures[29].

**Figure 4** High pressure leaks

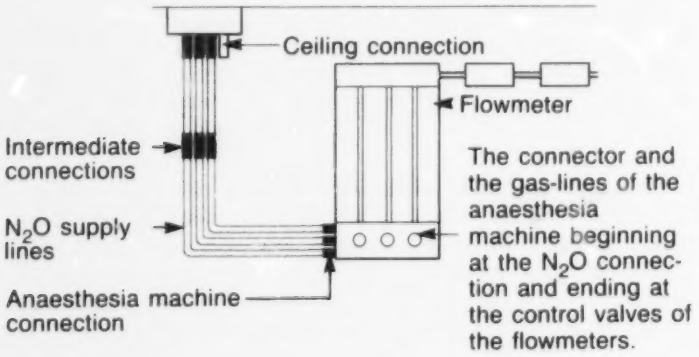
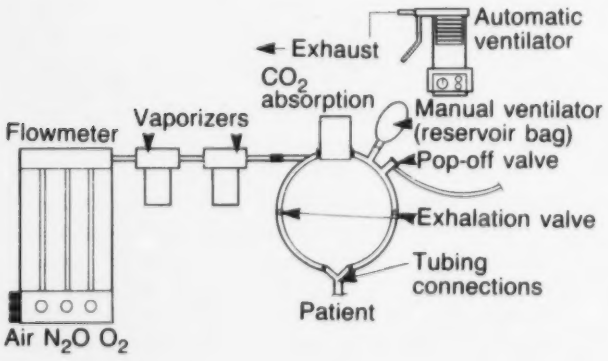

Ceiling connection
Flowmeter
Intermediate connections
$N_2O$ supply lines
Anaesthesia machine connection
The connector and the gas-lines of the anaesthesia machine beginning at the $N_2O$ connection and ending at the control valves of the flowmeters.

**Figure 5** Low pressure leaks




Leaks in the anaesthetic delivery system can be located by use of nitrous oxide detectors or by pressurizing the low pressure system and immersing in water. One schedule that has been suggested is the following:

Equipment maintenance *continued*

| Anaesthesia Equipment | Minimum Testing Frequency (months) | Method of Testing |
|---|---|---|
| Anaesthesia Machine (High Pressure) | 1 | Using nitrous oxide monitor, anaesthesia machine attached to gas supply. Flow meters- off. After 1 hour-level of $N_2O$ measured. Detectable levels on the monitor (greater than 5 ppm) represent leakage (with a non-recirculating air-conditioning system). |
| Anaesthesia Machine (Low Pressure) | 1 | Using $N_2O$ monitor check all potential sources of leakage (connections,-8f-absorber). Alternatively, soap solution can be used on connections. |
| Ventilators | 1 | Using $N_2O$ monitor, visual inspection of gaskets, etc. |
| Hoses and Bags | 4 | Using visual inspection for cracks, holes. Immersion in water. |
| Scavenging Tubing | 4 | Using visual inspections as above, immersion in water, or using $N_2O$ monitor. |

## Anaesthesia technique

Lecky and Springstead of the Department of Anaesthesia, University of Pennsylvania, have produced the following list of suggestions for techniques to substantially reduce anaesthetic spill[30]

1. Avoid turning on the $N_2O$ vaporizer until the mask is well fitted to the patient's face, or the patient is intubated and connected to the circuit. This ensures that all gas enters the scavenging system and room contamination is avoided.

2. Where possible, discontinue anaesthetic gas flows and empty the reservoir bag prior to suctioning or intubation. Again, this manoeuver prevents unopposed spill into the operating room environment.

3. At the end of a case, prior to extubation or removal of the mask, continue to administer $O_2$ alone as long as possible so that the scavenger system can eliminate the washed out anaesthetic gases. As well as benefiting the patient, this keeps spill to a minimum.

4. Exercise great care in filling vaporizers to avoid spills of volatile anaesthetic agents. Spills of small amounts of volatile agent result in markedly elevated levels in the operating theatre. Where possible, vaporizers should be filled after the daily slate of operations is complete. Any spillage is then removed by the ventilation system with no one exposed.

5. Maintain good mask fit which is critical. It is possible to run quite low levels during a case using a mask and assisted or controlled ventilation. With poor mask technique, however, high levels result. Often very small changes in mask angle can mean the difference between a good and a bad fit.

In addition, the use of low flow techniques can dramatically reduce contaminant levels in the operating theatre.

## General ventilation

Even with a well maintained scavenging system, small amounts of waste anaesthetic gas leak to the operating room, giving detectable concentrations. Further control can be applied through effective general room ventilation. This is also important in recovery rooms where general ventilation is the only effective way to dealt with anaesthetic gases present from the exhalations of patients in post-operative care.

The American Society of Heating, Refrigerating and Air-Conditioning Engineers (ASHRAE) has recommended in its standard for Ventilation for Acceptable Indoor Air Quality[33] that

General ventilation *continued*

for operating rooms the minimum fresh outdoor air requirements are 40 cfm/person occupying the room. For recovery rooms they recommend 15 cfm/person.

Monitoring

The effectiveness of the measures taken to reduce anaesthetic gases can be evaluated through regularly monitoring the operating room environment. Portable devices which measure pollutant levels by the absorption of infra-red radiation can be used to check instantaneous nitrous oxide levels. Average exposure to nitrous oxide can be obtained by measuring the instantaneous levels at specified times throughout the work day and calculating the average over this time period. As well, average exposure can be determined by collection of air samples over the work day in mylar bags using portable sampling pumps. The concentration of nitrous oxide is obtained by passing the collected air through the portable nitrous oxide monitor. Alternatively, the sampling pumps can be used to collect the nitrous oxide on adsorption devices filled with molecular sieve 5A as a trapping agent. Subsequently the nitrous oxide is desorbed by heat and its concentration determined by infra-red absorption or gas chromatography. Average concentrations of halogenated anaesthetics can be determined in a similar fashion, using activated charcoal as the trapping agent. Desorption of the halogenated agents from the charcoal is accomplished using heat or a solvent, and the concentrations are analyzed by gas chromatography or infra-red absorption.

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